Right-ear precedence and vocal emotion contagion: The role of the left hemisphere

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Abstract

Much evidence suggests that the processing of emotions is lateralized to the right hemisphere of the brain. However, under some circumstances the left hemisphere might play a role, particularly for positive emotions and emotional experiences. We explored whether emotion contagion was right-lateralized, lateralized valence-specifically, or potentially left-lateralized. In two experiments, right-handed female listeners rated to what extent emotionally intoned pseudo-sentences evoked target emotions in them. These sound stimuli had a 7 ms ear lead in the left or right channel, leading to stronger stimulation of the contralateral hemisphere. In both experiments, the results revealed that right ear lead stimuli received subtly but significantly higher evocation scores, suggesting a left hemisphere dominance for emotion contagion. A control experiment using an emotion identification task showed no effect of ear lead. The findings are discussed in relation to prior findings that have linked the processing of emotional prosody to left-hemisphere brain regions that regulate emotions, control orofacial musculature, are involved in affective empathy processing areas, or have an affinity for processing emotions socially. Future work is needed to eliminate alternative interpretations and understand the mechanisms involved. Our novel binaural asynchrony method may be useful in future work in auditory laterality.

Keywords:

Echo Suppression Precedence Effect Auditory Lead-Lag Interaural Time Difference Social Affect

Introduction

The involvement of the two hemispheres of the brain in the processing of aspects of emotion has been a topic of study for a considerable period of time, but there are still unresolved issues. One of these has been whether emotion is preferentially processed by the right hemisphere (Right Hemisphere Hypothesis, supported by e.g. Borod, Zgaljardic, Tabert, & Koff, 2001; Bryden, Ley, & Sugarman, 1982; Gainotti, 2012; Hugdahl, Iversen, & Johnsen, 1993; Ley & Bryden, 1982; Mitchell, Elliott, Barry, Cruttenden, & Woodruff, 2003; Schepman & Rodway, 2007; Schepman, Rodway, & Pritchard, 2016; Wildgruber, Pihan, Ackermann, Erb, & Grodd, 2002) or whether there are situations in which the left hemisphere may also play a role in processing emotions. For example, under the valence-specific laterality hypothesis, the right hemisphere, particularly in frontal regions, is more attuned to negative and the left to positive emotions (e.g. Borod, 1993; Davidson, 1984; Jones & Fox, 1992; Jansari, Rodway & Goncalves, 2011; Jansari, Tranel & Adolphs, 2000; Killgore, & Yurgelun-Todd, 2007; Rodway, Wright & Hardie, 2003; Tomarken, Davidson, Wheeler, & Doss, 1992; see also Najt, Bayer, & Hausmann, 2013, for a nuanced discussion). Similarly, under the approach-withdrawal hypothesis, which links emotion to motivational behaviours, the right hemisphere is specialized for the processing of emotions that may lead to the withdrawal from aversive stimuli, while the left hemisphere is specialized for the processing of emotions that may lead to the approach towards appetitive stimuli (e.g. Davidson, 2004; Harmon-Jones, 2004; see also Hecht, 2010). Another proposal is that, while the right hemisphere is dominant for the direct subjective experience of affect for primary emotions, the left hemisphere has a dominant role in social emotions (Ross, Homan, & Buck, 1994). Ross et al.'s proposal is based on their observations that patients reinterpreted emotional memories during right-hemisphere Wada tests, from primary emotions such as fear, sadness

and anger, to social emotions such as feeling embarrassed, silly, sorry, etc. More recently, but somewhat similarly, it has been proposed that the right hemisphere processes direct affect, while the left hemisphere may be selectively involved in the processing of the information conveyed by the emotional state for the purpose of directing higher level behaviours. This may include self-regulatory behaviours, such as reappraisal of emotions, or problem solving, and may contain elements of verbalization (Shobe, 2014).

Overall, a large body of evidence exists that supports lateralized processing of emotion in the right hemisphere. Further, there is a smaller but still substantial body of evidence that suggests potential involvement of the left hemisphere in emotion processing, but there seems to be less convergence in the data and theories regarding the conditions under which the left hemisphere becomes involved. Thus, an increasing number of researchers propose that the next important task for the field is to establish under which conditions the different hypotheses are supported empirically (e.g. Prete, Laeng, Fabri, Foschi, & Tommasi, 2015), which includes determining which type of emotional processing may involve which hemisphere.

Our focal interest in the current study was in emotion contagion (Hatfield, Cacioppo, & Rapson, 1994). Emotion contagion, particularly what is known as “primitive emotion contagion” (Hatfield, Cacioppo, & Rapson, 1992), is thought to occur automatically when people exposed to expressions of emotion produced by others show or experience the same emotion as the transmitter of the emotion. Emotion contagion can occur in response to relatively brief stimuli, though emotion contagion is also used to describe responses to events of longer durations. This contrasts with mood transfer, a term which tends to be reserved for

the induction of a mood via longer exposure to emotional material. Much work on emotion contagion has used facial stimuli, while some has used human non-verbal affective vocalizations (e.g. laughing vs. crying, Sestito et al., 2013), but our interest in the current study was in contagion via the prosody of emotional speech. Emotion transfer from a speaker's prosody to a listener has been relatively underexplored empirically, but Neumann and Strack (2000) found that exposure to happy / sad prosody led to mood transfer in listeners. More recently, it has even been found that listeners whose speech output was modified before being played back to themselves were affected in their own vocal emotions (Aucouturier, Johansson, Hall, Segnini, Mercadié, & Watanabe, 2016). The lateralization of vocal emotion contagion has also received relatively little attention. An important exception is work by Papousek, Reiser, Weber, Freudenthaler, & Schulter, (2012, who discovered higher emotional responsiveness as measured by electroencephalogram activation to nonverbal affective sounds (e.g. crying, laughing) in individuals with greater left than right dorsolateral activation at rest, potentially suggesting a left-hemisphere involvement in emotion contagion.

The current study builds on Schepman, Rodway and Geddes (2012), in whose discussion it was hypothesized that valence-specific laterality, which was observed in some circumstances in vocal emotion processing, may potentially be related to emotion contagion in listeners. Emotion contagion contains elements of both emotion perception and emotion experience, and it had been proposed previously that emotional experience may play a role in the emergence of emotion lateralization data compatible with valence-specific laterality, due to emotional experience triggering the activation of frontal cortical areas (Borod, 1993; Davidson, 1984; Tomarken et al., 1992). The current research builds on this idea, and an important aim of the current study was to examine whether, if frontal regions are involved in

emotional experience in a valance-specific way, then there may be valence specific lateralized emotion contagion. This formed the first of one of our three competing hypotheses for the study.

In the absence of prior work that addressed the lateralization of emotion contagion via prosody, we had to be cautious in our hypotheses. Thus, in acknowledgement of the fact that emotion contagion may start with emotion perception, it is possible that emotion contagion is lateralized to the right hemisphere, in line with the large body of evidence suggesting a right-hemisphere specialization for emotion perception, and which has been found very consistently for vocal stimuli (e.g. Godfrey & Grimshaw, 2016, 2015; Ley & Bryden, 1982; Mitchell et al., 2003; Rodway & Schepman, 2007; Wildgruber et al., 2002, 2005). Therefore, based on the body of work that places vocal emotion processing in the right hemisphere, right-lateralization of emotion contagion formed the second of our three competing hypotheses for the study.

A third alternative hypothesis is that emotion contagion may be left-lateralized. While, as discussed, the left hemisphere is typically less associated with emotion perception, it is possible that emotion contagion may be left-lateralized, potentially because the task might contain elements of interpretation (Shobe, 2014) or because mechanisms supporting affective flexibility may be located in the left hemisphere (Papousek et al., 2012). Thus, we take left-lateralization of emotion contagion as the third of our three competing hypotheses.

In our study, we examined whether experiential measures of emotion contagion showed evidence of lateralization. We operationalized our study with subjective ratings (1-9) of emotion evocation as the dependent variable. We used emotionally intoned pseudo-sentences (Banse & Scherer, 1996), so that there was no intelligible speech content in the stimuli, and only the suprasegmental and paralinguistic vocal cues (e.g. voice quality, pitch, tempo, and loudness) were informative to the listener.

Dichotic presentations, with different stimuli in the left and right channel, are commonly used to ensure presentation of auditory stimuli to the intended contralateral hemisphere. However, we felt that this was not suitable for a task in which emotion contagion was to be rated, as listeners would not necessarily readily empathize with a dichotic stimulus, and it would not resemble natural conditions. Thus, to present these stimuli laterally, we used a stimulus onset asynchrony of 7 ms in one ear (see also Rodway & Schepman, 2007, control stimuli). This causes a percept of the stimulus emanating from the temporally leading channel. This is a phenomenon known as the precedence effect (e.g. Zurek, 1987; Brown, Stecker & Tollin, 2015). The leading wave of multiple temporally separated sound waves is identified as representing the source of the sound directly, with lagging waves being interpreted as having been reflected off nearby surfaces and undergoing echo suppression. The suppression of echoes is thought to enhance the intelligibility and localization of the main signal (Cranford & Romerein, 1992; Litovsky, Colburn, Yost & Guzman, 1999). While there is still some uncertainty over the exact mechanisms that may contribute to this percept we argue, based on pre-existing evidence, that it leads to disproportionate stimulation of the hemisphere contralateral to the leading stimulus.

A brief overview of the anatomical structures that lead to a contralateral representation of auditory signals is needed first. In a summary description of the auditory pathways, Hugdahl (2000) explains that input from a particular ear travels as a neural signal up the auditory pathway via a series of relay stations. Of these, the projections from the third relay station, the lateral lemniscus, to the fourth relay station, the inferior colliculus of the tectum, are primarily contralateral, which ultimately leads to a stronger representation of the stimulus input in the auditory cortex contralateral to the stimulus input ear. The neural consequence of this anatomical arrangement can be observed at the cortical level with auditory evoked magnetic fields measured with magnetoencephalography, in which sounds played in an ear correspond to a peak in the contralateral hemisphere that has a shorter latency and a higher amplitude than the ipsilateral response (Nakasato, et al, 1995; Pantev, Ross, Berg, Elbert, & Rockstroh, 1998).

Research on the precedence effect has demonstrated that the precedence effect occurs reliably in humans and animals with interaural time differences in the range used by us (see Brown et al., 2015, for a recent review). There is some debate over the location of the mechanisms along the ascending auditory pathway that lead to the perceptual effects, which we discuss in outline below, but in its totality, the evidence suggests that due to echo suppression mechanisms, a lagging stimulus is suppressed, making its neural and perceptual representation weaker than a leading stimulus. If the leading stimulus is presented to one ear and the lagging stimulus to the opposite ear along the contralaterally arranged pathways, then the hemisphere receiving the leading stimulus from the contralateral ear will be stimulated disproportionately due to the combined effects of echo suppression and the anatomical arrangement of the pathways.

Much research has been conducted on the precedence effect, but there is not yet full consensus on the exact location of the mechanisms that contribute to the percept. There is evidence that the lagging wave is likely to undergo echo suppression, which increases the relative strength of the leading wave (see e.g. Yang & Grantham, 1997). A structure of key importance is the inferior colliculus, which contains neurons that are sensitive to lead and lag, as established by single-cell recordings in animals (e.g. Litovsky & Yin, 1998; see also Litovsky, Fligor, & Tramo, 2002, for a human neurological case study). It is suggested that more complex auditory-cortical mechanisms may also play an essential role in behavioural manifestations of the precedence effect (see e.g. Blauert & Braasch, 2005; Litovsky et al., 1999; Trahiotis & Hartung, 2002). A systematic study of the effect was carried out by Fitzpatrick, Kuwada, Kim, Parham, and Batra (1999), who measured the neural responses to click pairs with varying lead-lag asynchronies in structures in the ascending auditory pathways of cats and rabbits, namely the auditory nerve, anteroventral cochlear nucleus, superior olivary complex, inferior colliculus, and primary auditory cortex. They found evidence of the suppression of the lag stimulus all the way up the pathway, and the higher up the auditory pathway, the longer the lead-lag delay that would lead to suppression. However, others (Damaschke, Riedel, & Kollmeier, 2005) have argued and demonstrated that lower-level structures (i.e. the lateral lemniscus and its termination into the inferior colliculus) that feed into a key element of the auditory brainstem response (i.e. wave V) show neural representations reflecting both leading and lagging signals without evidence of suppression and with a time difference reflecting the actual lead-lag stimulus time difference. In contrast, they found that a single fused percept was in evidence in cortical auditory evoked potentials, as demonstrated in auditory cortical responses via Mismatch Negativity. Damaschke et al. argue for a higher-level location of the mechanism supporting the precedence effect on the

basis of this finding, yet many other findings are compatible with a mechanism in the inferior colliculus, with potential additional mechanisms at cortical level (see Brown et al. 2015).

There is some uncertainty over the exact mechanisms by which the precedence effect occurs. However, based on the existing neuroanatomical and echo suppression evidence, a reasonable working assumption is that the presentation of stimuli to two separate ears with an interaural time difference leads to the suppression of the lagging stimulus, rendering the leading stimulus presented in the opposite ear relatively stronger. Both stimuli travel in these states up the ascending auditory pathways to contralateral primary auditory cortices. This leads to the primary auditory cortex in the hemisphere contralateral to the ear receiving the leading stimulus being more strongly stimulated than the hemisphere contralateral to ear receiving the lagging stimulus. Thus, the hemisphere contralateral to the ear in which the sound is leading would be disproportionately stimulated by our desynchronized stimuli. Once a signal has travelled to the primary auditory cortex, speech-like sounds (regardless of intelligibility) also activate areas that are lateral and anterior to the primary auditory cortex (Scott & Johnsrude, 2003). A similar upward path from the auditory cortices to higher-level processing areas applies here as it does to dichotic techniques (Brancucci, et al., 2004; Hugdahl, 2000).

In the main experiments, we had two independent variables, namely ear lead and emotion. We used four separate emotions (anger, fear, happiness and sadness), as these are recognized relatively readily via vocal cues (Banse & Scherer, 1996), including by adolescents with or without autism (Brennand, Schepman & Rodway, 2012). We chose to use female participants only, because there is some evidence that they may be more likely to report emotion contagion (see e.g. Wild, Erb, & Bartels, 2001), giving us more scope to detect any laterality

patterns. The valence-specific laterality hypothesis would predict a significant interaction, such that negative stimuli (anger, fear, sadness) would be rated as evoking higher levels of listener emotions with a left ear lead, and the positive emotion of happiness with a right ear lead. On the other hand, if emotion contagion were lateralized in line with the right-hemisphere hypothesis, then we would expect all stimuli to be rated as evoking emotions in the listener to a greater extent with a left ear lead than with a right ear lead, without an interaction. The third competing hypothesis, in which the left hemisphere may be dominant in emotion contagion, would have higher evocation scores with a right ear lead than with a left ear lead, without an interaction between the two factors. We report two experiments using this design, preceded by a stimulus pre-test. Following these we also report Experiment 3, an emotion recognition experiment with the same independent variables, but with accuracy of recognition in a four-alternative forced-choice task as the dependent variable, which served as a control experiment.

Experiments

Method

The pre-test and Experiments 1, 2 and 3 were approved by the University of Chester's Department of Psychology's Ethics Committee, and complied with British Psychological Society ethical guidelines. Participants took part voluntarily, in return for participant credit if eligible, no reward if not eligible. In Experiment 2 payment was also offered.

Pre-test

The pre-test served as a method for selecting a subset of suitable stimuli from a larger set.

Participants

20 female self-reported right-handed female students from the University of Chester with self-reported normal hearing took part in the pre-test.

Materials

112 stimuli from the Banse and Scherer (1996) database were included in the pre-test. These represented seven emotions, three of which represented two intensities of the same emotion. There were 16 stimuli in each of the seven subsets (original German labels are in parentheses to avoid ambiguities in mapping to the original database): hot anger (heißer Arger), cold anger (kalter Arger), fear / anxiety (Angst), panic fear (panische Furcht), happiness (Freude), elation (überschäumende Freude), and sadness (Trauer). Note that sadness only had one intensity level in our pre-test. We deemed the more intense version of sadness from the database, namely despair (Verzweiflung), not prototypical of vocal sadness due to the cues to high arousal present in the vocal stimuli. As fully described in Banse and Scherer (1996), 6 male and 6 female professional German television actors portrayed each emotion on one of two pseudo-sentences using method acting, producing distinctive prosodic cue patterns for each emotion. The pseudo-sentences consisted of phonemes and phonotactics that are commonly encountered in major European languages and are transcribed as “Hätt sandig prong nju wentsie” and “Vi gott leich jean kill gos terr”.

Procedure

All pseudo-sentences were played in a random order to participants wearing Sennheiser HD201 headphones via a computer running e-Prime. In this and all the other experiments the headphones had been tested for balance using a sound level meter. Adapting the procedure used in Wild et al. (2001), participants were asked to imagine that they were meeting the person they could hear in a neutral social situation such as in a café or on a bus but without having direct communication with the person. They were asked, after hearing each stimulus, to indicate to what extent the emotion that the speaker was expressing was evoked in themselves (i.e. to what extent they were feeling that emotion themselves). In a deviation from Wild et al. (2001), who took responses to a range of emotion adjectives, we were interested in taking a response only for the target emotion, which was displayed to the participants before and after each trial. This was done to avoid the risk of diluting the potentially fleeting impact of the emotion on the listener's feelings, which could have occurred as they responded to a longer list of adjectives. This concern applied particularly to the main experiment, which examined laterality, and we felt it was important to match the conditions of the pre-test to those of the main experiment. Participants were told that there was no response time limit but that we were interested in their spontaneous reaction, so that they did not need to think for a long time about their response. Participants were told that there were no right or wrong answers and that we were interested in their actual feelings, based only on listening to the speaker.

Each trial started with an emotion label (angry, afraid, happy, or sad) displayed centrally for 500 ms. Following a 250 ms delay, the auditory pseudo-sentence was then played, followed by a response prompt which read: Evokes (emotion), with below this the rating scale "not at all 1 2 3 4 5 6 7 8 9 very strongly". Participants entered a response when seeing this prompt. No hand use instruction was given and participants had free choice which hand(s) /

finger(s) to use to enter the responses. A 500 ms inter-trial interval was used. Following the main block of stimuli, a further six stimuli were played from the elation category without participants needing to respond, to ensure that listeners who may have been affected by the most recently heard stimuli left the experiment in a positive mood, in line with British Psychological Society ethical guidelines. Debriefing consisted of a brief wellbeing check and a chance to find out in more detail about the research.

Selection of stimuli and evocation scores of selected items

A selection of 12 stimuli per emotion (combining both intensities where applicable) was made from the 112 pre-tested stimuli. We selected stimuli which attracted a mid to high evocation score in the pre-test, with the constraint that there were equal numbers of female and male speakers for each emotion, the two pseudo-sentences were spoken with the same frequency by male and female speakers, and in the overall stimulus set the mean evocation scores were matched as closely as possible across emotions, and did not differ significantly from each other. Full details of the evocation scores for each stimulus and an indication of the items selected are presented in the Appendix, as these may be useful to other researchers. Descriptive statistics for the evocation scores for the selected items were: Anger: M = 5.90 SD = .96); Fear: M = 5.88, SD = .67, Happiness: M = 5.87, SD = .93, Sadness: M = 5.91, SD = .65. The evocation scores did not differ on a 1 x 4 repeated-measures ANOVA with mean evocation scores for the 12 items per emotion per participant as the dependent variable, $F(3, 44) = .006$, $p = .99$.

Experiment 1

The aim of Experiment 1 was to examine the lateralization of emotion contagion, using an ear lead manipulation.

Participants

The participants were 72 self-reported right-handed female students from the University of Chester with self-reported normal hearing. They had not participated in the pre-test.

Materials

As stated, based on the pre-test scores, we had selected 12 stimuli for each emotion (anger, fear, happiness, sadness; see pre-test and Appendix). All selected stimuli were processed using Adobe Audition 3.0 so that each sentence had two stereo versions. In one version, the sound in the left channel started 7 milliseconds before the sound in the right channel ("left ear lead"). In the other version, this was vice versa ("right ear lead"), yielding a total of 96 stimuli, created as 16-bit, 22,500 Hz. .wav files.

Procedure

The procedure was identical to that of the pre-test. All 96 files were played to all participants.

Results

Evocation scores were calculated for each emotion (anger, fear, happiness, sadness) in each ear lead version (left, right) for each participant, and subjected to a 2 x 4 repeated-measures Analysis of Variance. The data yielded the descriptive statistics in Table 1.

===== INSERT TABLE 1 ABOUT HERE =====

The key main effect of interest was that of ear lead, and this was significant, $F(1, 71) = 7.96$, $p = .006$, $\eta_p^2 = .101$. Averaging across the four emotions, evocation score was higher with a right ear lead (5.45) than a left ear lead (5.35). While this difference was subtle numerically, it was statistically robust. The effect of emotion was also significant, $F(2.7, 191.8) = 4.00$, $p = .011$, $\eta_p^2 = .053$ (with Greenhouse-Geisser correction due to a violation of sphericity). Despite matching at pre-test, some Bonferroni-corrected pairwise contrasts were significant in this larger sample of different participants. Anger led to lower evocation scores than fear ($p = .019$) and sadness ($p = .012$), with no other contrasts being significantly different. Here, too, the differences in the means that reached significance were relatively small numerically. In relation to valence-specific lateralization of emotion processing, the interaction between emotion and ear lead was not significant, $F(3, 213) = 1.33$, $p = .266$, $\eta_p^2 = .018$.

To give an indication of the time course of the responses, the median response time across all trials was 1354 ms. We did not analyze response latency data formally as a function of our independent variables, because speed of response was not relevant to the hypotheses.

Because there is an inherent concern that subjective ratings may not be as reliable as more objective measures, or that participants are able to respond randomly without detection of that random responding being possible, we ran correlational analyses to assess the test-retest reliability of these ratings. First, we took the average rating per pre-test item that was also used in the main experiment, and correlated it with the average ratings for the same item in the main experiment, collapsing over both ear lead conditions. The reader is reminded that these ratings were provided by different samples of participants. This yielded a very high Pearson correlation coefficient, $r = .836$, $N = 48$, $p < .001$. In addition, because each stimulus was rated twice by the participants in the main experiment, we ran a correlation between the average ratings for the left and right ear lead for each item. This yielded an even higher correlation, $r = .978$, $N = 48$, $p < .001$. Both these analyses suggest that, although the ratings were subjective, they were stable across different samples of listeners, as well as within the same listener across a shorter period of time, suggesting that they are reliable. The scatter plots accompanying both correlations are in Figure 1.

===== INSERT FIGURE 1 ABOUT HERE =====

Discussion of Experiment 1

The data showed that a right ear lead gave rise to reports of subtly but significantly stronger emotion contagion in comparison to a left ear lead, without a significant interaction. At first blush, this seems compatible with left-lateralization of emotion contagion, because, as we have argued, the hemisphere contralateral to the lead would be stimulated disproportionately. We must, however, consider alternative explanations. First of all, an important consideration will be addressed in Experiment 2. A right-ear lead may direct attention to the right of visual space, and therefore subtly move participants' responses to the right on the ascending number line used to collect the emotion contagion ratings. This would mean that our effect of ear lead was merely an artefact of a subtle shift of spatial attention, and not associated with the lateralization of emotion contagion. To examine whether such an interpretation could account for our findings, we ran a further study, in which we counterbalanced the anchors placed at the ends of the response scale. If the orientation of the number line were to be responsible for the effect in Experiment 1, then the effect should disappear with this counterbalancing.

Experiment 2

Method

Materials and Procedure

These were identical to Experiment 1, with the exception that there were two variants of the response scale. One variant was as before ("Original"), and one had the anchors reversed, so that 1 meant "very strongly" and 9 meant "not at all" ("Reversed").

Participants

We tested a further 95 participants who had not taken part in the pre-test or in Experiment 1, but who had the same characteristics as set out for Experiment 1. Participants were allocated to one of two variants of the response scale (explained in the following subsection) based on order of arrival. A slight imbalance in participant numbers arose due to the use of two research assistants and a minor participant numbering error during testing (which was subsequently corrected). This meant that there were 49 in the "Original" and 46 participants in the "Reversed" scale anchor placement variants. Participants in this study received £5 alongside their credits (if eligible for credits).

Results and brief discussion of Experiment 2

Data for the "original" scale were entered into the analysis as provided by participants, while data for the scale with reversed anchor placements were reversed back to be equivalent to the original scale for the purpose of analysis, before we combined both sets of scores. This means that in our reported data, the higher the evocation score was, the more self-reported emotion contagion was declared by the participants.

In this analysis, ANOVA factors were as for Experiment 1, but we added a between-subjects factor for scale anchor placement (Original, Reversed). Descriptive statistics in Table 2 show that, by and large, the higher levels of emotion contagion with a right ear lead were observed again, except in response to fear, where they were in the opposite direction for the "original" scale only, with no differences when the data for the two scale anchor placement conditions were combined.

===== INSERT TABLE 2 ABOUT HERE =====

With key relevance to our main hypothesis, the analysis showed a significant main effect of ear lead, $F (1, 93) = 7.64$, $p = .007$, $\eta_p^2 = .076$. Again, the means differed subtly (left ear lead: 5.76, right ear lead: 5.84), but robustly. This effect of ear lead did not interact significantly with that of scale anchor placement, $F (1, 93) = .12$, $p = .73$, $\eta_p^2 = .001$, or emotion, $F (3, 279) = 1.27$, $p = .29$, $\eta_p^2 = .013$, nor was it subject to a significant three-way interaction with emotion, $F (3, 279) = 1.31$, $p = .27$, $\eta_p^2 = .014$.

By way of background effects, there was a significant effect of emotion, as also observed in Experiment 1, $F (3, 279) = 12.39$, $p = .002$, $\eta_p^2 = .053$, and this effect interacted significantly with that of scale anchor placement, $F (3, 279) = 5.20$, $p < .001$, $\eta_p^2 = .118$. The main effect of scale anchor placement was not significant, $F (1, 93) = .61$, $p = .44$, $\eta_p^2 = .007$. In relation to the significant interaction, an inspection of the means showed subtly higher evocation scores for the original than the reversed scale anchors for all emotions except for sadness, where the opposite occurred. It is not clear why this pattern may have occurred, and, because it did not interact with the effect of ear lead, we will not discuss it further, as attempts at interpretation would be speculative.

The results from Experiment 2 replicated those from Experiment 1 while ruling out a spatial orientation explanation for the effect of the right ear lead. However, the results do not address further outstanding issues, because it is possible that the right ear lead (and assumed left-hemisphere involvement) associated with our emotion contagion task may simply be a by-product of the emotion perception aspects that may form part of the task. We therefore ran

Experiment 3 as a control experiment, with the aim of investigating whether the right lead advantage for emotion contagion observed in Experiments 1 and 2 would also be associated with a more traditional perception experiment. In addition, a perception experiment would allow us to observe whether in providing emotion evocation data participants are simply reporting the ease of perception of the emotion in the stimuli, or whether we may have reason to infer that emotion contagion is distinct from emotion perception.

Experiment 3

In this experiment, we wanted to establish whether the right ear lead advantage for emotion contagion would also be observed in a basic emotion perception task. If this were so, then the effect of ear lead observed in Experiments 1 and 2 could be a basic perceptual effect.

Method

Participants

There were 67 participants who had not taken part in any other studies, and had the same characteristics as for Experiments 1 and 2.

Materials and Procedure

The stimuli and their randomized order and the headphones were identical to those used in Experiments 1 and 2. As for the procedure, the participants were instructed to listen to the stimuli and identify after each stimulus whether they had heard Anger, Fear, Happiness or Sadness. They entered their answer in response to a question mark that appeared centrally

after each stimulus, accompanied by a reminder of the response options nearer the bottom of the screen, arranged in vertical list with one emotion per line, centred vertically: A = Anger, F = Fear, H = Happiness, S = Sadness. Responses were entered using the keyboard keys 6, t, g and b, respectively, on a traditional qwerty keyboard (marked with stickers A, F, H and S for the four emotions, respectively), chosen for the vertical orientation of their array, to avoid left-right orientations forming a confound in the study. A 500 ms fixation cross started each trial, while a 250 ms delay occurred between each fixation and the onset of the sound stimulus.

Results and brief discussion of Experiment 3

Mean proportions correct were calculated for each participant for each emotion and ear lead (see Table 3 for overall means), and these were entered into a 4 (emotion) x 2 (ear lead) repeated-measures ANOVA, which revealed that there was no significant main effect of ear lead, $F(1, 66) = .006$, $p = .94$, $\eta_p^2 = .00008$, and that ear lead did not interact with emotion, $F(3, 198) = .133$, $p = .27$, $\eta_p^2 = .020$. There was a significant main effect of emotion, $F(3, 198) = 74.51$, $p < .001$, $\eta_p^2 = .53$. An inspection of the means showed that there were very high accuracy levels for sadness, with lower but still considerably above 25% chance recognition levels for the other three emotions. Bonferroni-corrected pairwise comparisons showed accuracy for sadness differing significantly in accuracy from all the other emotions (all $p < .001$), and also accuracy for Anger differing significantly from accuracy for Fear ($p = .014$). In all, the data showed no effect of ear lead.

To explore the notion that the evocation scores might simply and solely reflect the ease with which participants could identify the emotions in the stimuli, we again ran a by-items correlation analysis, this time of the pre-test evocation scores for the relevant stimuli, against the mean proportion correctly identified for that same stimulus, averaging over all participants across both ears. This correlation was strongly positive and significant, $r = .502$, $N = 48$, $p < .001$. However, the correlation was weaker than that between the evocation scores in the pre-test and in Experiment 1, which, as reported above, was .836. The difference between these two correlation coefficients was significant on a two-tailed Fisher r-to-z transformation, $Z = 3.11$, $p = .0019$. This suggests that, while the evocation ratings correlate with the ease of the perception of the emotions in the stimuli, evocation scores are not simply and solely a reflection of the emotion recognition process, and may include additional processes, else the correlations would be not be expected to differ significantly.

We did not find a left-ear-lead advantage for this emotion recognition task, which might have been expected if the right hemisphere were disproportionately stimulated by the left-ear-leading stimuli, in light of previous findings of right-lateralisation for emotion recognition (e.g. Borod, Zgaljardic, Tabert, & Koff, 2001; Bryden, Ley, & Sugarman, 1982; Gainotti, 2012; Hugdahl, Iversen, & Johnsen, 1993; Ley & Bryden, 1982; Mitchell, Elliott, Barry, Cruttenden, & Woodruff, 2003; Schepman & Rodway, 2007; Wildgruber, Pihan, Ackermann, Erb, & Grodd, 2002). It is possible to hypothesise that this may mean that the manipulation is not successful in stimulating the contralateral hemisphere, but we feel that, based on the existing data on auditory pathways and direct measurements of echo suppression cited in the Introduction, that this is not the most plausible interpretation. There might be other reasons for this null result, such as different effects in opposite directions neutralizing each other. It is difficult to be certain without further experiments. For the purpose of our main focus of interest, which is the lateralization of emotion contagion, the finding in Experiment 3

suggests that the effects found in Experiments 1 and 2 seem task-dependent, and not solely stimulus-driven.

General Discussion

The data showed that a right ear lead, which we argued leads to a stronger left hemisphere stimulation, gives rise to reports of subtly but significantly stronger emotion contagion in comparison to a left ear lead, without a significant interaction with emotion. This is not compatible with right-hemisphere lateralization of emotion contagion, nor is it compatible with valence-specific laterality for emotion contagion. Instead, it is compatible with a left-hemisphere dominance for emotion contagion. This appears to be the first report of a left-hemisphere lateralization of emotion contagion via prosodic-affective aspects of the voice, but builds on work showing a link between emotion contagion and the left hemisphere (e.g. Papousek et al., 2012). In the discussion, we evaluate the plausibility of our interpretation against alternative explanations. This discussion also synthesizes existing evidence, including neuropsychological evidence from patients, and observations from electroencephalography and neuroimaging evidence to suggest mechanisms by which this effect might occur, which may serve to suggest areas for future research. However, we acknowledge the need for caution in the interpretation of this finding and a need for more empirical data to understand this effect in more depth. We discuss alternative explanations and cautionary notes first, before suggesting potential left-hemisphere brain regions in which mechanisms that may play a role in the observed effect could be located.

First, we need to acknowledge that our working assumption that the leading ear disproportionately stimulates the contralateral hemisphere would benefit from being supported by more direct evidence that observes activation patterns in key regions in the two hemispheres in response to the same stimuli that we used in our study. This is in part because the literature on the precedence effect is complex, and often uses simpler stimuli, such as click pairs instead of more complex speech signals (see Schwartz, Harris, & Principe, 1999). Future data may come from experiments using Magnetoencephalography, Functional Magnetic Resonance Imaging, or potentially Cortical Auditory Evoked Potentials. These are outside the scope of our current work, but could provide crucial evidence on the mechanisms of the precedence effect, specifically in the context of using ear leads in future auditory laterality research. Such evidence could increase the methodological repertoire for the investigation of important outstanding research questions about the lateralization of auditory processes that are difficult to investigate using established methods such as dichotic listening, and this would therefore be valuable.

Second, we need to acknowledge that the right ear-lead advantage and likely left-lateralization could potentially be triggered by linguistic elements of the experiment, rather than by emotion contagion. Firstly, the display of target words on the screen as part of the trial sequence might account for the effect, as such linguistic stimuli might activate the left hemisphere as part of the overall trial. However, in other work (e.g. Rodway & Schepman, 2007) words were also displayed on the screen and this did not lead to a left lateralization, which makes such an interpretation less immediately obvious. The pseudo-linguistic nature of the stimuli themselves may have led to a left-lateralization. However, if the effect were solely due to the nature of the stimuli, we would have expected a similar effect in Experiment 3, but this was not observed. The findings of Experiment 3 suggest that the right-ear lead advantage

for emotion contagion observed in Experiments 1 and 2 was task-dependent. In addition, other studies have used linguistic stimuli, and these do not automatically lead to left-hemisphere lateralization, but interact with the task (e.g. Ley & Bryden, 1982). Despite this, a fuller test of any potential role of the linguistic elements of the task could use different visual displays (e.g. face icons, manikins, or symbols). Thus, while the presence of linguistic elements in the trial sequence may be responsible for left-lateralization, they do not seem to be able to account fully for the data, both current and prior. This suggests that we may need to look for an explanation that features the emotion contagion itself as contributing to the effect.

Third, rating data can be inherently difficult to interpret, and it is not clear exactly what features listeners used to reach their rating decisions. One plausible interpretation could be that, when listeners rated the extent to which the emotion expressed by the speaker was evoked in themselves, they rated whether the emotion was expressed well, or even simply whether the emotion was recognizable. However, for such an explanation to hold fully, one would expect emotion recognition data to correlate very strongly with emotion contagion data, and we found that this was not the case. The correlation between the pre-test emotion evocation data and the emotion recognition data was significantly weaker than that between the pre-test data and the emotion contagion data from Experiment 1. This suggest that, while emotion recognition may feed into the process of emotion contagion, emotion contagion does not seem to be exactly the same as emotion recognition.

An interpretation that emotion contagion via emotional prosody may involve the left hemisphere demands the identification of plausible potential mechanisms in the left hemisphere that might underlie this effect neurally. We briefly discuss four potential types of

mechanisms, namely affective flexibility, motor mimicry, affective empathy, and social processing, each of which have evidence locating key components in the left hemisphere. Although our current data cannot distinguish between these interpretations, they might provide useful avenues for further research.

First, our interpretation that emotion contagion may be left-lateralized chimes with work by Papousek, et al (2012), who, as briefly discussed in the Introduction, showed that participants with greater left than right activation in the dorsolateral frontal cortex at rest were more responsive in their electroencephalogram signal when listening to affective vocalizations (laughing and crying). Further work (Papousek, Weiss, Schulter, Fink, Reiser, & Lackner, 2014), with audiovisual material (film clips) suggests that the finding, at least for negative material, was not restricted to affective vocalizations, as it was also observed for individuals watching horrifying scenes in which people were injured (e.g. car crashes). The mechanism proposed is an emotion regulation mechanism located in the dorsolateral frontal cortex. It is possible that our findings are linked to such a mechanism, but further work would be needed to explore this.

Second, there is evidence to suggest that as well as patients with right-hemisphere damage, there are also some patients with left-hemisphere damage, particularly around the left frontal operculum, who show impairments in emotion recognition from prosody (Adolphs, Damasio, & Tranel, 2002). The orofacial musculature is neurally represented in this region, and it is possible that neural representations of the orofacial muscles play a role in emotion perception via the voice. Other researchers have demonstrated that patients with damage to the left frontal operculum show specific deficits in prosodic abilities, particularly in relation to the

production, articulation and initiation of speech (Alexander, Naeser & Palumbo, 1990). Moreover, the condition known as buccofacial apraxia (e.g. Woolley, 2003) evidences a link between the left frontal operculum and the expression of emotion. In addition, it is also noted that areas around the left frontal operculum are innervated by direct projections from the limbic system (Alexander et al. 1990, Freedman, Alexander, & Naeser, 1984). This may provide a fast route for vocalizations motivated by emotions such as alarm calls and could lend plausibility to a link between emotion perception, emotion experience, and vocal production (see Sauter & Eimer, 2010). Thus, an interpretation around motor area activation in the left hemisphere that forms part of the processing of the vocal emotional speech signal may form an explanation of our findings. However, this explanation comes with some reservations, as we will now discuss.

This line of evidence links neural motor representations to the perception of auditory speech. The role of neural activation of motor-cortical areas in the perception of vocal sounds has been extensively debated in relation to phonemic speech sounds. Following early proponents of this view (Liberman, Cooper, Shankweiler, & Studdert-Kennedy, 1967; Liberman & Mattingly, 1985), a more recent revival has been associated with mirror neurons (e.g. Di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992; Fadiga, Craighero, Buccino, & Rizzolatti , 2002; Galantucci, Fowler, & Turvey, 2006; Heyes, 2010; Iacoboni, 2008; Meister, Wilson, Deblieck, Wu, & Iacoboni, 2007). However, the role of motor activation in speech perception has been questioned, for example based on the retained ability of patients with Broca's aphasia to perceive speech (Hickok, 2010; Lotto, Hickok, & Holt, 2009), as has the role of mirror neurons, which have not been found in speech areas (e.g. Mukamel, Ekstrom, Kaplan, Iacoboni, & Fried, 2010). Thus an interpretation in which our finding is linked to a

motor-speech interface in the left hemisphere must be considered tentative until there is clearer evidence to consolidate it.

Third, more recently, a different area in the left hemisphere's cortex, the left inferior frontal gyrus, has been observed to be involved in both the production and perception of emotional signals, with the activation in these regions correlating with affective empathy (Aziz-Zadeh, Sheng, & Gheytanchi, 2010). It has also been shown (Shamay-Tsoory, Aharon-Peretz, & Perry, 2009) that the affective (as opposed to cognitive) element of empathy is associated with this region, based on patients with damage to Brodmann area 44, who showed specific emotional empathy deficits, while those with cognitive empathy deficits had lesions localized to the ventromedial prefrontal cortex. Similarly, Nummenmaa, et al. (2008) showed a similar pattern of left-lateralization when viewers were instructed to empathize with people depicted in emotionally evocative images (e.g. of an attack), but not when they empathized cognitively with people in neutral scenes. Nummenmaa et al. found that, in contrast to cognitive empathy, emotional empathy increased brain activity in ten clusters, which were primarily located in the left hemisphere. Further, Ochsner, et al., (2004) identified greater activation of the left inferior lateral prefrontal cortex when viewers judged emotions of others in affective images, as opposed to judging their own feelings in response to images, or engaging in a neutral control judgement. Thus, this set of studies potentially links the frontal regions of the left hemisphere to emotion contagion via brain regions involved in general affective empathy. Again, an interpretation along such lines may be plausible, but must remain tentative until further evidence is available.

A fourth plausible mechanism for the left-lateralization of emotion contagion is based on Ross et al.'s (1994) notion that the right hemisphere may be specialized for primary affect, but that the left hemisphere may be more involved in social aspects of emotional processing. As emotion contagion arguably has a social dimension, this could also be a reason why emotion contagion could be left-lateralized. Ross et al. (1994) found that the left hemisphere may become dominant during emotion processing when the emotion is not just felt at a basic level, but also has to be processed at a social level. Ross et al. (1994) observed that with the right hemisphere temporarily anaesthetized during the Wada test, patients recalled emotional memories in such a way that social emotions (embarrassment etc.) displaced the more primary emotions (e.g. fear, anger) that they had included in their pre-Wada recall of the same event, with factual aspects of the memories unaffected. More recent research by Shamay-Tsoory, Lavidor, and Aharon-Peretz, (2008) has confirmed this lateralization pattern in the visual domain, with complex emotions being left-lateralized and basic emotions right-lateralized. They showed this in patients with left- or right-hemisphere prefrontal cortical damage (but without significant language impairments), and separately in unimpaired participants. Taken together, these studies may support an interpretation that emotion contagion processing is left-lateralized and that a potential reason may be that social processing of emotions is left-lateralized. Again, we cannot be certain until further empirical work has been done.

In conclusion, we propose that we have found, for the first time, evidence that emotion contagion via emotionally intoned pseudo-sentences is stronger with a right ear lead, and may therefore be left-lateralized. We linked this, tentatively pending further investigation, to potential plausible mechanisms in the left hemisphere. In the absence of more precisely localized data (e.g. from neuroimaging studies), we cannot be certain how the observed effect

is caused, and more direct evidence would need to be obtained to understand this effect in more depth. Further investigation may also strengthen our understanding of our novel method for future use in auditory laterality research.

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Appendix

The stimuli were selected from a database entitled Geneva Vocal Emotion Expression Stimulus Set (GVEESS). It can be obtained via the Geneva Emotion Research Group http://www.unige.ch/cisa/gerg.html at the Centre Interfacultaire en Sciences Affectives (CISA) at the University of Geneva.

In the current Appendix, stimuli with their pre-test evocation scores based on 20 participants (within-subjects) who used a scale of 1 – 9 to indicate to what extent the emotion portrayed was invoked in themselves, with 1 indicating “not at all” and 9 “very strongly”.

In the Table, the Stimulus column reflects the original file names from the Banse and Scherer (1996) database. Quoted from information accompanying the database, and made applicable to the subset of files used in our study: “The file names provide the essential information about each stimulus: First letter: emotion: anxiety = A(ngst), happiness = F(reude), hot anger = H(eiβer Arger), cold anger = K(alter Arger),) panic fear = P(anische Furcht), sadness = T(rauer), elation = U(berschäumende Freude). First number: one of two scenarios provided to the actors: 1 = scenario 1; 2 = scenario 2. Second number: sentence used: 1 = Hätt sandig prong nju wentsie; 2 = Vi gott leich jean kill gos terr. Third number: repetition (actors produced each stimulus twice): 1 = first portrayal; 2 = second portrayal. Fourth number: actor/speaker identification number 01-12.” Note that speaker sex is reflected in the Table. The column marked “Emotion” indicates the emotion displayed on the screen in both pre-test and main experiment. For the column “Selected”, 0 = not selected, 1 = selected for the main study.

Stimulus	Emotion	Speaker's Sex	Sentenc e	Evocation score	Selected
H11108.wav	anger	male	1	6.40	1
H11203.wav	anger	male	1	7.60	0
H11209.wav	anger	female	1	5.70	1
H11211.wav	anger	female	1	7.45	0
H12112.wav	anger	male	2	7.80	0
H12203.wav	anger	male	2	7.10	1
H12209.wav	anger	female	2	7.30	0
H12211.wav	anger	female	2	6.85	1
H21111.wav	anger	female	1	6.05	1
H21203.wav	anger	male	1	7.90	0
H21207.wav	anger	female	1	5.05	1
H21212.wav	anger	male	1	7.40	1
H22105.wav	anger	female	2	6.45	1
H22109.wav	anger	female	2	6.35	0
H22112.wav	anger	male	2	7.30	0
H22203.wav	anger	male	2	7.20	0
K11103.wav	anger	male	1	4.80	0
K11111.wav	anger	female	1	3.50	0
K11207.wav	anger	female	1	1.85	0
K11212.wav	anger	male	1	2.30	0
K12108.wav	anger	male	2	4.35	0
K12111.wav	anger	female	2	4.50	0
K12203.wav	anger	male	2	4.50	1
K12209.wav	anger	female	2	5.10	0
K21108.wav	anger	male	1	4.80	1
K21202.wav	anger	female	1	2.60	0
K21204.wav	anger	male	1	3.55	0
K21209.wav	anger	female	1	3.30	0
K22111.wav	anger	female	2	4.20	0
K22203.wav	anger	male	2	5.05	1
K22204.wav	anger	male	2	3.00	0
K22209.wav	anger	female	2	5.40	1
A11103.wav	fear	male	1	4.40	0
A11202.wav	fear	female	1	3.25	0
A11203.wav	fear	male	1	3.55	0
A11205.wav	fear	female	1	4.15	0
A12111.wav	fear	female	2	2.70	0
A12112.wav	fear	male	2	3.15	0
A12202.wav	fear	female	2	3.45	0
A12203.wav	fear	male	2	4.75	0
A21202.wav	fear	female	1	3.85	0
A21203.wav	fear	male	1	5.80	1
A21206.wav	fear	male	1	1.85	0
A21207.wav	fear	female	1	4.00	0

A22101.wav	fear	male	2	3.20	0
A22103.wav	fear	male	2	5.40	0
A22202.wav	fear	female	2	2.50	0
A22207.wav	fear	female	2	2.35	0
P11103.wav	fear	male	1	4.70	0
P11110.wav	fear	female	1	4.95	1
P11203.wav	fear	male	1	7.00	1
P11210.wav	fear	female	1	3.90	0
P12111.wav	fear	female	2	3.75	0
P12203.wav	fear	male	2	6.75	1
P12205.wav	fear	female	2	5.75	1
P12212.wav	fear	male	2	5.10	0
P21202.wav	fear	female	1	5.40	1
P21203.wav	fear	male	1	6.10	1
P21204.wav	fear	male	1	5.05	0
P21210.wav	fear	female	1	4.85	1
P22105.wav	fear	female	2	5.80	1
P22111.wav	fear	female	2	5.40	1
P22112.wav	fear	male	2	6.25	1
P22203.wav	fear	male	2	6.50	1
F11104.wav	happiness	male	1	2.90	0
F11111.wav	happiness	female	1	5.15	0
F11203.wav	happiness	male	1	3.00	0
F11211.wav	happiness	female	1	3.95	0
F12107.wav	happiness	female	2	3.85	0
F12112.wav	happiness	male	2	4.40	0
F12202.wav	happiness	female	2	3.30	0
F12212.wav	happiness	male	2	4.10	0
F21102.wav	happiness	female	1	4.45	0
F21103.wav	happiness	male	1	4.75	1
F21203.wav	happiness	male	1	3.80	0
F21209.wav	happiness	female	1	3.35	0
F22104.wav	happiness	male	2	3.25	0
F22205.wav	happiness	female	2	5.20	0
F22211.wav	happiness	female	2	5.45	0
F22212.wav	happiness	male	2	4.25	0
U11103.wav	happiness	male	1	6.65	1
U11107.wav	happiness	female	1	6.70	1
U11211.wav	happiness	female	1	6.60	1
U11212.wav	happiness	male	1	7.90	0
U12109.wav	happiness	female	2	4.95	1
U12111.wav	happiness	female	2	6.85	1
U12112.wav	happiness	male	2	5.80	1
U12208.wav	happiness	male	2	3.75	0
U21103.wav	happiness	male	1	4.45	0
U21108.wav	happiness	male	1	5.30	1
U21111.wav	happiness	female	1	5.65	1
U21202.wav	happiness	female	1	4.90	0
U22107.wav	happiness	female	2	6.25	0
U22203.wav	happiness	male	2	4.50	1

U22211.wav	happiness	female	2	5.40	1
U22212.wav	happiness	male	2	7.30	1
T11111.wav	sadness	female	1	4.15	0
T11112.wav	sadness	male	1	7.40	0
T11204.wav	sadness	male	1	5.25	1
T11205.wav	sadness	female	1	5.65	1
T12104.wav	sadness	male	2	5.30	1
T12105.wav	sadness	female	2	6.55	1
T12205.wav	sadness	female	2	6.20	1
T12212.wav	sadness	male	2	7.10	1
T21109.wav	sadness	female	1	6.35	1
T21203.wav	sadness	male	1	5.85	1
T21204.wav	sadness	male	1	5.70	1
T21209.wav	sadness	female	1	5.95	1
T22103.wav	sadness	male	2	6.35	1
T22202.wav	sadness	female	2	4.10	0
T22203.wav	sadness	male	2	7.50	0
T22209.wav	sadness	female	2	4.70	1

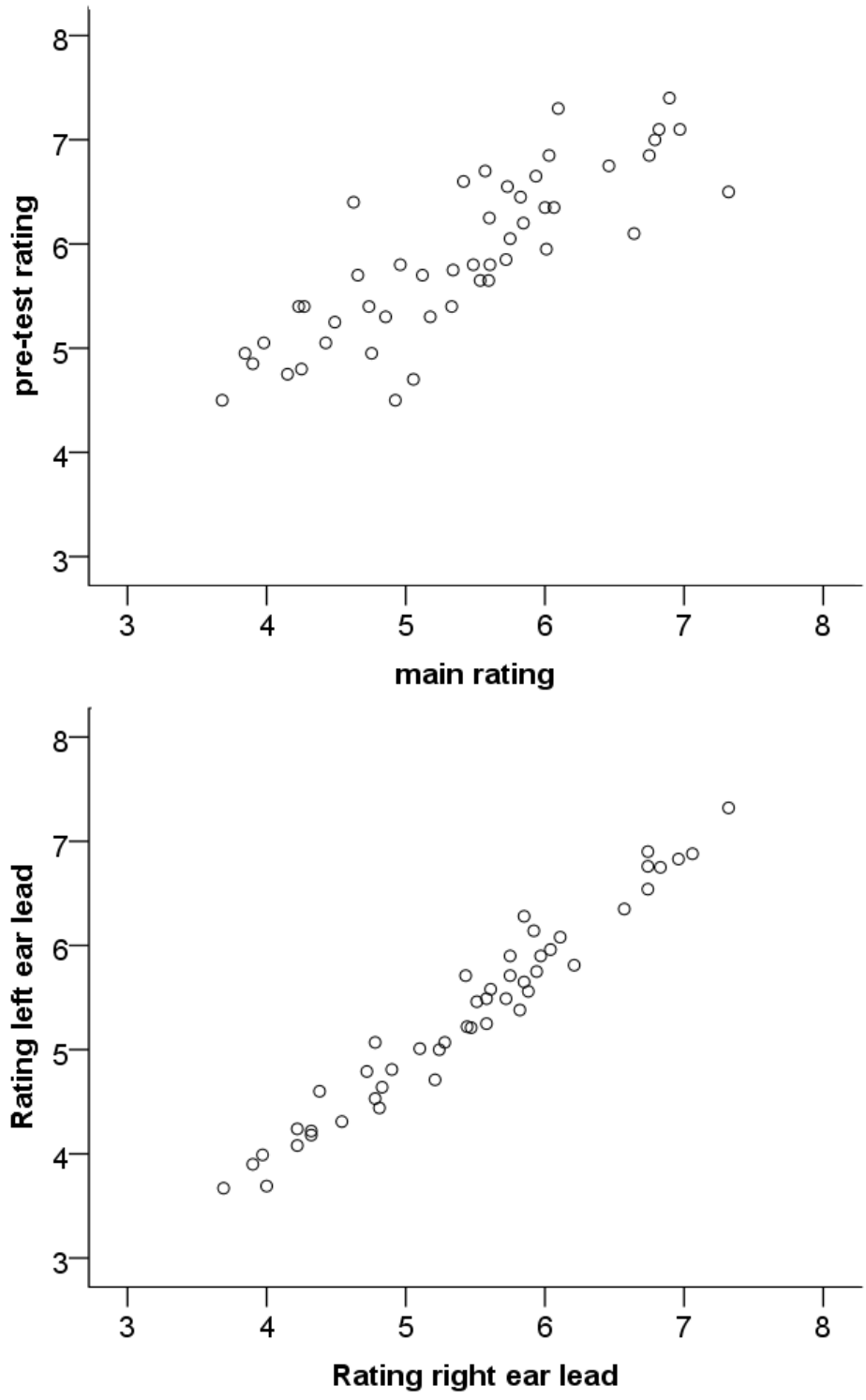

Figure 1: Scatter plots with the top panel representing pre-test ratings against main experiment ratings (collapsing over both ear leads), and the bottom panel representing the ratings in the main experiment with the left and right ear lead.

Table 1

	Left ear lead		Right ear lead	
	Mean	SD	Mean	SD
Anger	5.11	1.76	5.22	1.75
Fear	5.35	1.70	5.56	1.60
Happiness	5.36	1.67	5.38	1.70
Sadness	5.57	1.74	5.63	1.75

Table 1: Means and Standard Deviations for emotion evocation ratings as a function of emotion and ear lead for the main experiment, on a 1-9 scale (1 = not at all, 9 = very strongly).

Table 2

		Left ear lead		Right ear lead	
		Mean	SD	Mean	SD
Original	Anger	5.69	1.35	5.78	1.44
	Fear	6.16	1.25	6.10	1.20
	Happiness	5.66	1.13	5.86	1.12
	Sadness	5.89	1.61	5.98	1.58
Reversed	Anger	5.29	1.67	5.39	1.80
	Fear	5.87	1.42	5.93	1.41
	Happiness	5.17	1.44	5.23	1.55
	Sadness	6.29	1.50	6.46	1.57
Total	Anger	5.50	1.52	5.59	1.62
	Fear	6.02	1.34	6.02	1.30
	Happiness	5.42	1.30	5.55	1.38
	Sadness	6.08	1.56	6.21	1.58

Table 2: Means and Standard Deviations for emotion evocation ratings as a function of emotion, ear lead and scale anchor placement for Experiment 2. Following reverse-scoring, all ratings are expressed here on an ascending 1-9 scale with 1 = not at all, 9 = very strongly.

Table 3

	Left ear lead		Right ear lead	
	Mean	SD	Mean	SD
Anger	0.68	0.15	0.67	0.15
Fear	0.60	0.17	0.63	0.17
Happiness	0.67	0.17	0.65	0.17
Sadness	0.91	0.11	0.90	0.11

Table 3: Means and Standard Deviations for accurate identification of emotions in Experiment 3 (proportions).